ISSN: 2581-5989
PubMed - National Library of Medicine - ID: 101738774

International Journal of Dental Science and Innovative Research (IJDSIR)

IJDSIR : Dental Publication Service

Available Online at: www.ijdsir.com

Volume – 3, Issue – 6, November - 2020, Page No. : 107 - 113

Ogilvie's Syndrome a Rare Cause of Colonic Obstruction- Case Series and Review of Literature.

[1]Shivraj singh chauhan, Head consultant, Department of Surgery, Tata main hospital, Jamshedpur- 01, India

[2]Niranjan Kumar, Associate specialist, Department of Surgery, Tata main hospital, Jamshedpur- 01, India.

[3]Shuvam chandi prasad pati, Post graduate (DNB), Department of Surgery, Tata main hospital, Jamshedpur- 01, India

Corresponding Author: Niranjan Kumar, Associate specialist, Department of Surgery, Tata main hospital, Jamshedpur- 01, India.

Citation of this Article: Shivraj singh chauhan, Niranjan Kumar, Shuvam chandi prasad pati "Ogilvie's Syndrome A Rare Cause of Colonic Obstruction- Case Series And Review of Literature. ", IJDSIR- November - 2020, Vol. – 3, Issue - 6, P. No. 107 – 113.

Type of Publication: Review Article

Conflicts of Interest: Nil

Abstract

Ogilvie's syndrome is an uncommon condition where acute pseudo-obstruction of the colon, characterized by dilatation of colon occurs in the absence of any underlying mechanical obstruction. The condition is deemed to be due to an imbalance in the autonomic nervous system (sympathetic overactivity and parasympathetic suppression). It usually presents in the 7th decade with slight male preponderance. The diagnosis of Ogilvie's syndrome is based on clinical and radiological findings. The treatment is usually non-operative and the modalities include conservative treatment, drug treatment with injection neostigmine, and colonoscopic decompression. Surgical management is reserved for non-responders or those, who develop complications or signs of peritonitis. The objective of this study is to re-emphasize the clinical presentation and radiological findings to establish a diagnosis and initiate treatment without delay. We reviewed five consecutive cases of Ogilvie's syndrome managed by us over the last 5 years. These cases present a common spectrum of management, where all described treatment modalities, varying from non-operative treatment to surgical intervention were used.

Keywords: Acute colonic pseudo-obstruction, Ogilvie's syndrome, Neostigmine, Decompression.

Introduction

Ogilvie's syndrome which is also termed Acute colonic pseudo-obstruction (ACPO) was first described by Sir William Heneage Ogilvie (British surgeon) in 1948 [1]. It is defined as acute dilatation of the colon in the absence of an anatomic lesion that obstructs the flow of intestinal contents. ACPO is an uncommon cause of intestinal obstruction usually occurring in admitted patients with severe coexistent comorbid conditions, which is associated with increase morbidity and mortality [2,3]. Most cases can be managed non-operatively, however surgery is the treatment of choice in complicated cases. Timely recognition is the most important key in the

management of patients to decrease mortality and morbidity. The mortality rate documented for medical management is 14%, whereas it is 30-40% in surgically treated patients [3]. We share our experience on the presentation and management of five cases of ogilvie's syndrome to draw attention of this rare condition.

Case Presentation

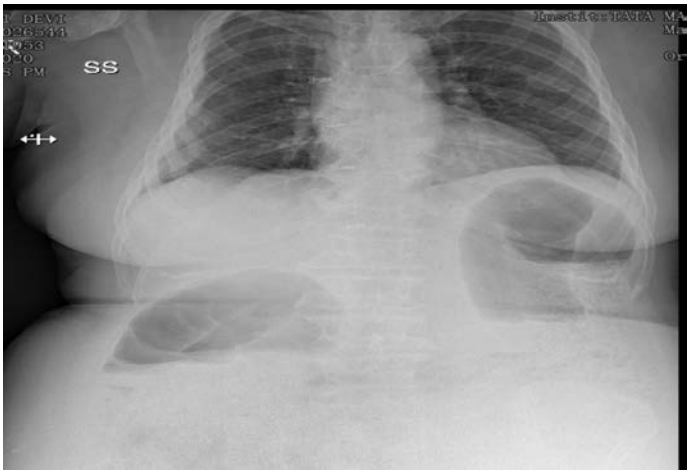

Figure 1a: X-ray of abdomen showing isolated dilated colonic loop.

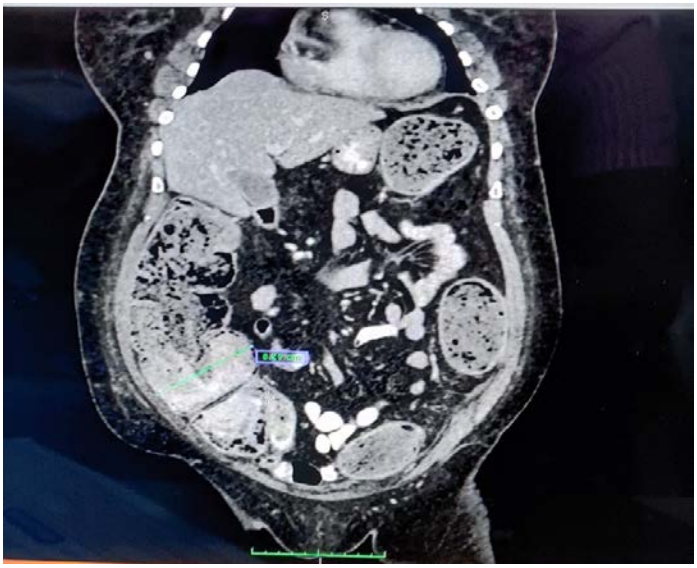

Figure 1b: CECT abdomen showing isolated dilated colon with caecum diameter 8.29cm.

Case No	Age, sex	History & examination	Co-morbidity	Blood investigation & radiological finding	Management	Result
1.	66Y/F	Abdominal discomfort & fullness-1week Obstipation- 3days. On Examination (O/E)- vital stable, Bowel sounds (BS)-increased Per abdomen(P/A)-soft, gaseous distention, non-tender Per-rectal(P/R)-normal	Diabetes (DM) Hypertension (HTN), Hypo-thyroidism	K-3.1meq/l, Na-133meq/l X-ray abdomen -dilated large bowel, normal small bowel (Figure-1a). CECT abdomen- gross isolated colonic-dilation with fecal loading without any mass lesion or lumen narrowing, caecal diameter-8.29cm (Figure-1b,1c)	Conservative	-Abdomen girth decreased 90cm from 102cm. -flatus passed on 4th day. -Discharge on 7th day.

2.	60Y/M	Abdominal discomfort, distension & obstipation - 2days O/E- Vital stable, BS-Normal P/A- diffusely distended with no guarding, rigidity & rebound tenderness or mass abdomen. P/R- normal	DM	Urea-60mg/dl, createnine-2mg/dl. Rest blood parameter unremarkable. X-ray abdomen- grossly dilated colon, with few air-fluids. CT of abdomen- massive colonic-dilatation, caecal diameter 9 cm without any mass lesion or stricture.	Conservative	- flatus Passed on 3rd day. -Discharge on 5th day.
3.	72/M	Abdomen discomfort, distension & obstipation-4 days. O/E- Vital stable, P/A- distended, deep tenderness of right iliac fossa. BS-N	BPH IHD	Unremarkable blood investigation. X-ray of abdomen-isolated dilatation of large bowel. CT of abdomen–dilatation of colon without any obstructive lesion, caecal diameter 8 cm, Suspicious sigmoid asymmetric wall thickening noted s/o? fecal residue /??mass lesion.	Conservative ↓ (on 3rd day) Sigmoidoscopy for assessing wall thickening & to decompress (no mass, fecal residue +) ↓ Flatus tube placed (colonoscopic guided).	Copious drainage in flatus tube, 2 day later flatus tube remove. -Distension resolved, -Discharge on 8th day
4.	76Y/F	Discomfort & distension of abdomen-10days Obstipation & anorexia -5days H/O Gastrectomy 30 days back. Pt was bed bound since surgery O/E- vital stable, P/A- healed scar of previous surgery with	HTN	K-2.9meq/lt, Na-130meq/lt (at admission) K-3.6meq/l, Na-139meq/l (on 2nd day) X-ray of abdomen-dilated large bowel with few air fluid. CT of abdomen-Grossly	Conservative treatment x 2 days escalated to Drug injection started on 3rd day bolus dose of neostigmine	-Passed flatus freely on 4th day after 2nd dose of neostigmine [within 24 hrs of

		grossly distended abdomen, Non-tender, BS - sluggish		dilatated colon [caecal diameter 8 cm] without any obstructive lesion /zone of transition	(2mg IV over 30 min) Two dose given-on 3rd and 4th day.	neostigmine] - distention relieved - discharge on 8th day.
5.	85Y/ M	Distension of abdomen, obstipation -12 days. O/E- Mostly bed bound,Vital stable, P/A-gross distension, soft, nontender, no guarding mass abdomen.	DM	Unremarkable blood parameter. CT of abdomen-gross entire colonic dilatation [caecal diameter 8 cm] from caecum to rectum without mass or narrowing.	Conservative x2 days (failed)→ Neostigmine on 3rd day→ (failed) developed ↑abdominal distension, tachypnea→surgical decompression & Caecostomy.	-Moved Stoma on POD -3 -tolerated orally from POD 4 -Discharge on POD 10 ,14th day of admission.

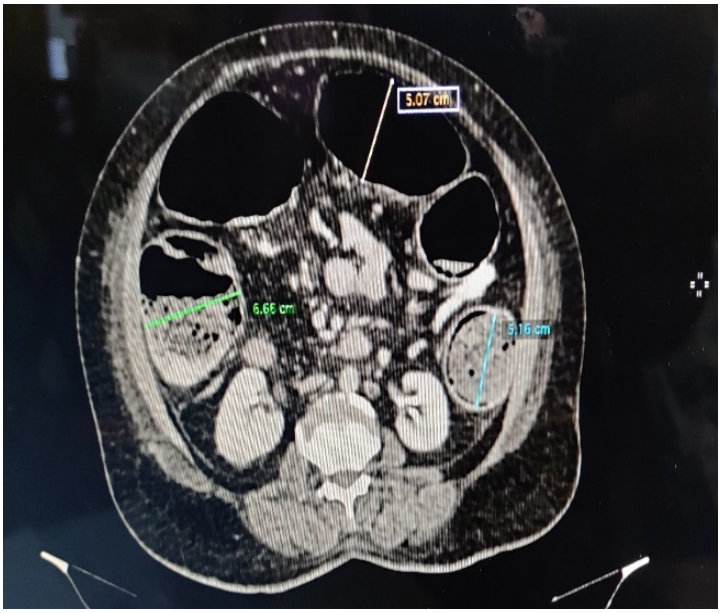

Figure 1c: CECT of abdomen showing dilated large bowel loops.

Discussion

In Ogilvie's syndrome, there is dilatation of colon due to failure of peristaltic wave progression in large bowel [functional], without any mechanical obstruction. The highest prevalence is observed in late middle age (mean age 60years) with slight male predominance (60%) [4]. In 1986, Vanek et al reported the mean age of 56.5years for males and 59.9years for female patients in cases of ACPO [3]. Clarke et al studied 30 patients of Ogilvie syndrome, and found male to female patient ratio of 3:1. The mean age of patient was 74.3years. In our study mean age in male & female patients was 70.6 year and 71year respectively. The disease usually involves caecum and right colon, but it can involve any part or even entire colon [5]. According to Laplace law, part of the colon which has the greatest diameter, is thinnest and most likely to perforate, hence caecum is the most common site of perforation. There are multiple predisposing factors and clinical conditions responsible for ACPO, these are as follows — trauma (especially fractures), burns, major surgery in recent past (obstetrical, pelvic, major orthopaedic surgery, abdominal or cardiothoracic surgery), severe medical illness (pneumonia, myocardial

infection, or heart failure), cardiac disease, neurological condition, retroperitoneal pathology (such as malignancy or haemorrhage), metabolic imbalance, medication (e.g. opioids, narcotics), and idiopathic [3,6].

In our study, old age [7th decade onwards], surgery in recent past, DM, hypothyroidism and metabolic derangements were found to be associated with ACPO. Ogilvie's syndrome is a rare complication of surgery, that occurs in 0.06% patients after cardiac surgery, 0.29% of burn patients and 0.7-1.3% of patients after orthopaedic surgery [7,8]. The exact pathogenesis of acute colonic pseudo-obstruction is not completely understood. But the current theory states that it results from the imbalance of the autonomic nervous system. In general, the parasympathetic stimulation increases gut mobility, whereas the sympathetic nervous system decreases gut mobility and constrict the sphincters. Derangements in the autonomic nervous system either by sympathetic over activity or parasympathetic suppression or a combination of both results in colonic atony and causes pseudo-obstruction. The patient of Ogilvie's syndrome usually presents with diffuse abdominal pain with distension (80%) (Not colicky pain), nausea & vomiting, (60%), obstipation (60%) [4]. Vanek et al [3] and Grassi et al found that abdominal distension is the most common finding in the case of ACPO. Metabolic abnormalities, especially hypokalemia, hypocalcemia, and hypomagnesemia, are common, occurring in more than 50 percent of patients [3].

The management of ACPO depend on early diagnosis and assessment for the signs of impending / actual colonic perforation. We followed the guideline by the American society for Gastrointestinal Endoscopy and clinical practice from American society of colon and Rectal Surgeons to treat ACPO [9,10]. Which includes- conservative management with observation, drug treatment with injection neostigmine, colonoscopic decompression, Surgical colonic decompression (by creating stoma / resection of ischaemic/perforated colon, in those developing peritoneal signs / non-responders). There is some evidence that neostigmine treatment given after colonoscopic decompression, may shorten hospital stay. In one of our cases, colonoscopic done before neostigmine to rule out suspicious lesion in colon, which was found in of CT of abdomen. The diagnosis of acute colonic pseudo-obstruction is based on history, clinical examination and radiological findings. Clinical examination shows no palpable mass or features of dynamic bowel obstruction. Plain X-ray abdomen shows isolated dilated colon with few air fluid levels. If mechanical colonic obstruction is suspected, a water-soluble contrast enema or CECT scan of the abdomen should be performed to differentiate it from mechanical obstruction. The sensitivity and specificity of contrast enema to diagnose Ogilvie syndrome is 96% and 98% respectively [11], whereas for CECT scan the sensitivity and specificity are 91% each [12]. CECT scan offers additional advantages over the contrast enema i.e. finding accurate measurement of bowel diameter, detecting bowel ischemia and necrosis of bowel. Perforation most commonly occurs when the ceacal diameter is more than 14cm (up to 23%) [3]. There is evidence of increased risk of colonic perforation with caecal diameter greater than12cm [13]. The duration of dilation greater than six days corroborate better than caecal diameter alone in predicting perforation.

On conservative treatment, 83-96% of patients with uncomplicated Ogilvie's syndrome resolve within 2-6days of the initiation of treatment [14], making it the first-line of therapy. Conservative management includes nasogastric tube (for bowel rest), rectal tube placement (for decompression), Correction of electrolyte imbalance,

discontinuation of narcotics, and treatment of underlying cause and infection [7]. Endoscopic colonic decompression, was described by Kulkora and Dent in 1977, is the second line of management in patient who fail to respond to conservative treatment or in whom neostigmine is contraindicated. If colonoscopy shows signs of colonic ischemia, then the procedure must be interrupted and laparotomy planned. The colonoscopic decompression procedure is difficult and potentially dangerous in these patients, because of the following reason [7]:

A. Unprepared bowel hampers visibility, gentle saline/tap water enema can improve vision
B. Time-consuming procedure for a sick patient, requires almost 40-60minutes
C. There is a risk of perforation due to air insufflation in already dilated bowel.
D. Hindrance in evaluation of colonic mucosa, to look for signs of ischemia due to unprepared bowel.

Drug management with injection Neostigmine has emerged as an efficacious treatment for Ogilvie syndrome, not resolving with conservative management. Neostigmine is a reversible cholinesterase inhibitor, which increases acetyl choline availability and stimulates muscarinic receptors of colon leading to increase in colonic motor activity [15]. 2milligram Injection neostigmine is administered slowly intravenously over 3-5minutes. Positive response (passing flatus and stool and decrease in abdominal distension) can result within 30 min of injection. However, in both of our cases, where neostigmine was used, flatus was passed after 3 hrs. It can be repeated if there is an incomplete response or there is recurrence. In our case too 2nd dose of Neostigmine was required. The second dose has proven effective in 40-100% of patients. The second dose of neostigmine is administered after an interval of 80min which is the half-life of drug [15]. Neostigmine administration can have serious side effects which include – (a) GI side effects (salivation, nausea, vomiting, abdominal pain), (b) Cardiovascular effects (bradycardia, hypotension), (c) Respiratory (bronchospasm). Hence Neostigmine administration should be done under medical supervision and cardiac monitoring. Injection atropine is administered, if there is severe bradycardia. To reduce the side effects of Neostigmine, IV infusion @ (0.4-0.8 mg/hr over 24hrs) rather than bolus administration is preferred. Decreasing the dose of bolus to 1milligram instead of 2milligram too can reduce side effects. In both our patient's bolus dose of 2mg over 30 min was used. Contraindications to use of neostigmine are as follows:

Absolute contraindications: - colonic ischemia and/or perforation, uncontrolled cardiac arrhythmias, severe bronchospasm.

Relative contraindications- bradycardia, asthma, COPD, renal insufficiency (serum creatinine > 3 mg/dl), patient already on beta-blockers and myocardial infarction. Ponec et al. reported first randomized study in 1999 in which Injection neostigmine was used. In this study 10 (91%) out of 11 who received neostigmine resulted in prompt colonic decompression.

Surgical intervention is reserved for the following situations: - 1). Failure of respond to non-operative treatment. 2). Caecal diameter >14 cm. 3). Sign of bowel perforation or ischemia. Chudzinski et al. in 2015 suggested colostomy or cecostomy as the damage control procedure, for treatment of ACPO. In our study of five cases, two cases were managed conservatively, one case was relieved by colonoscopic decompression and one patient required two doses of IV neostigmine. While one of five patients (who was non-responder to non-operative management and worsened clinically) underwent laparotomy.

Conclusion

Ogilvie's syndrome is a rare entity that is characterized by acute dilatation of colon, without any evidence of mechanical obstruction. Increasing colonic distention >12 cm can lead to colonic ischemia and perforation. The first line of management is conservative treatment followed by medical treatment (IV Injection Neostigmine) or colonoscopic decompression. Surgery becomes the only option, in case of the presence of peritonitis.

References

1. Ogilvie H. Large-intestine colic due to sympathetic deprivation. British Medical Journal. 1948 Oct 9;2(4579):671.

2. Kaiser AM. Ogilvie transition to colonic perforation. The American Journal of Surgery. 2010 Jul 1;200(1): e15-6.

3. Vanek VW, Al-Salti M. A cute pseudo-obstruction of the colon (Ogilvie's syndrome). An analysis of 400 cases. Dis Colon Rectum. 1986 Mar. 29(3):203-10. [Medline]

4. Kadesky K, Purdue GF, Hunt JL. Acute pseudo-obstruction in critically ill patients with burns. The Journal of burn care & rehabilitation. 1995 Mar 1;16(2):132-5.

5. Jacob SE, Lee SH, Hill J. The demise of the instant/unprepared contrast enema in large bowel obstruction. Colorectal Disease. 2008 Sep;10(7):729-31.

6. De Giorgio R, Barbara G, Stanghellini V, et al. The pharmacological treatment of acute colonic pseudo-obstruction. Alimentary pharmacology & therapeutics. 2001 Nov 6;15(11):1717-27.

7. Johnston G, Vitikainen K, Knight R, et al Changing perspective on gastrointestinal complications in patients undergoing cardiac surgery. The American journal of surgery. 1992 May 1;163(5):525-9

8. Norwood MG, Lykostratis H, Garcea G, Berry DP. Acute colonic pseudo-obstruction following major orthopaedic surgery. Colorectal Disease. 2005 Sep;7(5):496-9.

9. Vogel JD, Feingold DL, Stewart DB, et al. Clinical practice guidelines for colon volvulus and acute colonic pseudo-obstruction. Diseases of the Colon & Rectum. 2016 Jul 1;59(7):589-600.

10. Naveed M, Jamil LH, Fujii-Lau LL, et al. American Society for Gastrointestinal Endoscopy guideline on the role of endoscopy in the management of acute colonic pseudo-obstruction and colonic volvulus. Gastrointestinal Endoscopy. 2020 Feb 1;91(2):228-35.

11. Chapman AH, McNamara M, Porter G. The acute contrast enema in suspected large bowel obstruction: value and technique. Clinical radiology. 1992 Oct 1;46(4):273-8.

12. Beattie GC, Peters RT, Guy S, Mendelson RM. Computed tomography in the assessment of suspected large bowel obstruction. ANZ journal of surgery. 2007 Mar;77(3):160-5.

13. Jayaram P, Mohan M, Lindow S, Konje J. Postpartum Acute Colonic Pseudo-Obstruction (Ogilvie's Syndrome): A systematic review of case reports and case series. European Journal of Obstetrics & Gynecology and Reproductive Biology. 2017 Jul 1;214: 145-9.

14. Mac Call C, Mac Cannell KL, Baylis B, Less S et al. Treatment of acute colonic pseudo-obstruction with cisapride, Gastroenterology 1990:98:773-6.

15. Saunders MD, Kimmey MB. Evidence-based gastroenterology and hepatology 2nd ed.Oxford; Blackwell Publishing;2004: PP.303-309.